AMENDMENTS TO THE CLAIMS

1-26. **(Canceled)**

27. **(Currently Amended)** A method for reducing memory dysfunction associated with damaged hippocampal tissue in a mammal, comprising ~~contacting a hippocampal cell with~~ administering to the mammal a morphogen comprising a conserved C-terminal seven-cysteine skeleton that is one or more of the following:

(a) at least about 60% identical to residues 330-431 of human OP-1 (SEQ ID NO: 2); and

(b) at least about 70% homologous to residues 330-431 of human OP-1 (SEQ ID NO: 2),

thereby reducing memory dysfunction associated with damaged hippocampal tissue in the mammal.

28. **(Previously Presented)** The method of claim 27, wherein said morphogen stimulates synapse formation between hippocampal neurons.

29. **(Previously Presented)** The method of claim 28, wherein said morphogen comprises residues 30-292 of SEQ ID NO:2.

30. **(Previously Presented)** The method of claim 28, wherein said morphogen comprises residues 330-431 of SEQ ID NO:2.

31. **(Previously Presented)** The method of claim 28, wherein said morphogen comprises residues 48-292 of SEQ ID NO:2.

32. **(Previously Presented)** The method of claim 28, wherein said morphogen comprises the amino acid sequence of SEQ ID NO:2.

33. **(Canceled)**

34. **(Currently Amended)** The method of claim 28, wherein said morphogen comprises residues ~~292~~ 293-431 of SEQ ID NO:2.

35. **(Previously Presented)** The method of claim 28, wherein said morphogen comprises residues 30-431 of SEQ ID NO:2.

36. **(Previously Presented)** The method of claim 28, wherein said morphogen is a BMP-2 polypeptide.

37. **(Previously Presented)** The method of claim 28, wherein said morphogen is a BMP-5 polypeptide.

38. **(Previously Presented)** The method of claim 28, wherein said morphogen is a BMP-6 polypeptide.

39. **(New)** The method of claim 27, wherein the hippocampal tissue damage is caused by a neurotoxin.

40. **(New)** The method of claim 27, wherein the hippocampal tissue damage is caused by transient global ischemia.

41. **(New)** The method of claim 27, wherein the hippocampal tissue damage is caused by permanent global ischemia.

42. **(New)** The method of claim 27, wherein the hippocampal tissue damage is caused by

Traumatic Brain Injury (TBI).

43. (**New**) The method of claim 27, wherein the morphogen is administered by intraventricular administration.

44. (**New**) The method of claim 27, wherein the morphogen is disposed in a biocompatible microsphere.

45. (**New**) The method of claim 27, wherein the damaged hippocampal tissue is damaged from mechanical trauma, chemical trauma, glucose deprivation or a neurotoxin.

46. (**New**) The method of claim 27, wherein the neurotoxin is selected from ibotenic acid, lead, ethanol, ammonia and formaldehyde.

47. (**New**) The method of claim 46, wherein the neurotoxin is lead.

48. (**New**) The method of claim 27, wherein the mammal is afflicted with senility, malnutrition, glucose metabolism disorder, or anorexia.

49. (**New**) A method for reducing memory dysfunction associated with damaged hippocampal tissue in a mammal, comprising administering to the mammal a morphogen comprising a conserved C-terminal seven-cysteine skeleton that is one or more of the following:

(a) at least about 60% identical to residues 330-431 of human OP-1 (SEQ ID NO: 2); and

(b) at least about 70% homologous to residues 330-431 of human OP-1 (SEQ ID NO: 2),

wherein the hippocampal tissue damage is caused by a neurotoxin,

wherein the neurotoxin is lead,

wherein the morphogen is disposed in a biocompatible microsphere,

thereby reducing memory dysfunction associated with damaged hippocampal tissue in the

mammal.

50. (**New**) The method of claim 49, wherein the mammal is afflicted with senility.